FOREIGN POULTRY DISEASES

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All over the world the poultry industry is undergoing vast changes, moving from small back year flocks to very large flocks of highly bred, high-producing stock which are reared in virtual isolation on artificial diets. There large flocks are extremely susceptible to diseases introduced from outside, and with the enormous increase in the traffic of poultry and poultry products, there has been a steady rise in the number of nation-wide outbreaks of poultry diseases in many foreign countries, generally attributed to the movement of infected breeding stock, eggs, chicks, etc. In the past, disease outbreaks overseas were generally isolated and scattered over the country and were mainly due to pullorum disease, fowl pox and

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coccidiosis, fowl typhoid and intestinal parasites. The institution of pullorum testing in the United Kingdom, however, greatly reduced the incidence of this disease in British birds exported to overseas countries, many of which now show much less pullorum than formerly. Routine vaccinations for fowl pox and the use of improved anthelmin-thics and coccidiostatic drugs have largely contributed to the improved control of these diseases in many overseas territories. The general disease picture abroad is therefore rather different from that of 20 years ago. The installation of large modern poultry houses with the common fault of chilling through drafts or over-heating with stuffy atmospheres, has led to the emergence of many epizootics of virus respiratory diseases, and with the increase in deep litter units has come an increase in diseases such as keratomalacia from irritant ammonia vapor, colds through the inhalation of dust, and intestinal bacterial and viral infections from dirty litter. Overseas it has been observed that fowl leucosis on deep litter occurs twice as often as it does on semi-intensive range units. New diseases are also emerging overseas, particularly avian encephalomyelitis and infectious sinusitis, no doubt due to overcrowding and the increased stress conditions found in modern poultry production methods. Other diseases

appear to be increasing in incidence such as Newcastle Disease, chronic respiratory disease, and the fungal diseases.

There is no doubt that the introduction of modern methods in Africa and Asia is enormously increasing production, but it would appear that the former losses from infectious diseases are to a certain extent being replaced with losses from stress conditions such as cage paralysis, cannibalism, feather-eating, bullying, etc., even though the premises are clean, warm, and comfortable and the food is adequate. It is interesting to observe that, despite adequate housing and food, somewhat similar stress conditions coupled with lack of exercise appear to be causing an increase of cardiac troubles, gastric and intestinal ulcers and neuroses in the human race.

In under-developed countries, the poultry disease picture is of course very much affected by malnutrition in the majority of flocks encountered. Animal protein is generally in short supply and the ignorant peasant owner will either underfeed his birds or he will feed stale fermented feed or grains which are deficient in vitamin A. The general picture is therefore one of retarded growth, poor hatchability, nutritional encephalomalacia, curl toe paralysis and coryza.

On the brighter side, modern methods overseas are greatly reducing the losses from intestinal worms, particularly where birds are kept on dry, clean litter for the first three weeks of age and then on wire floors until eight weeks of age. This reduction in losses can also be attributed to the improved anthelminthics now available to peasant farmers, the improved knowledge of life cycles enabling the introduction of strategic treatment regimes and the increasing use of mass treatments by water-soluble drugs and by micronized dust guns, instead of dosing each bird individually. In passing, I would mention that veterinary research workers appear to be on the verge of a break-through with a vaccine from irradiated worm larvae for the prevention of lung-worm in cattle. If successful in cattle, the technique may have some promise in the production of similar vaccines against poultry worms.

In many foreign countries the worst scourges of poultry were the insect-borne diseases such as spirochetosis, Egyptianellosis, etc. These are now being controlled by cheap insecticides like pyrethium and gammexane, which are now readily available for peasant farmers. However, we must not forget that despite all the extra weapons available to control poultry diseases, the most important

measures on the premises are still regular cleansing and disinfection and de-population.

In recent years, there has been a tendency for poultry owners overseas to rush into using drugs for a quick cure without fully realizing the consequences. Deaths may be quickly stopped but chicks, apparently cured, have grown up into adult carriers excreting infected, sometimes drug resistant organisms (fowl typhoid). Other birds may be cured but actually sterilized of infection which leaves them with no immunity and therefore liable to reinfection (coccidiosis). Veterinarians overseas have a tremendous task in educating poultry owners to realize that drugs should not be judged purely on their ability to reduce mortality. Other reasons for caution in the use of drugs are the risks of lowering flock fertility, delaying growth, and even killing healthy chicks and poults (blackhead in turkeys). As with drugs, vaccines should only be used after the careful consideration of the factors involved. Some vaccines interfere with diagnostic tests, some produce excretors of living virus organisms, and others can actually spread egg-transmitted diseases. In this connection it is advisable to use duck-embryo egg vaccines rather than avianized vaccines if possible.

In many countries overseas, the control of poultry disease through the restriction of movement of poultry and their products is practically impossible for economic and administrative reasons. It is noticeable how often disease is spread from hatcheries and producing plants as in the case of South Africa where epidemic tremor spread rapidly until hatcheries were brought under strict control. In passing, it can be observed that several chick diseases can now be controlled at hatcheries by dipping the warm hatching eggs in antibiotic drugs before they are shipped. American experience is that antibiotic egg-dipping is impracticable under present commercial conditions. In the case of Newcastle Disease, unboiled waste containing poultry scraps and ships waste in particular are particularly dangerous and most countries now have orders for boiling waste fed to poultry and hogs. It is often forgotten that poultry epizootics have often started from the uncontrolled traffic in infected parrots and pigeons and there appears to be a need everywhere for stricter legislation over the cage-bird industry. It is the author's personal opinion that the poultry industry overseas stands in the presence of a continuous threat from this source of

disease. In Africa numerous outbreaks are frequently caused by the custom of presenting a live bird as a gift to visiting guests.

There are no reliable figures to show whether the actual mortality from poultry disease in the world today is relatively higher than 20 years ago, but there is no doubt that losses in poultry and production have increased in importance in the terms of the national economy. In the past, veterinarians have paid little attention to poultry diseases and have, in fact, tended to ignore such low-grade infections as infectious bronchitis. Nowadays infectious bronchitis in Britain is known to be capable of causing a 20 per cent reduction in egg production with a further 20 per cent of the remaining eggs rendered unmarketable. This is as serious as some of the major killing diseases from the poultryman's point of view. For this reason veterinarians must give increased attention to low-grade poultry diseases. In the USA broiler plants alone are estimated to have an overall mortality of 5 per cent, being as high as 30 per cent in some areas, representing a national average annual loss of $47 per 100 birds, from disease alone. The picture is probably worse overseas. With the development of the quick-freeze techniques and with the possibility of viruses

remaining infective in bone-marrow and muscle for many months, the potential threat of low-grade infections is likely to increase steadily.

The modern poultryman overseas receives more attention from veterinarians now than in the past. An interesting example occurred in 1961 when sudden and severe outbreaks of hepatitis were reported in many flocks in East Africa and Britain at about the same time. The cause, originally presumed to be a virus, was rapidly narrowed by veterinary researchers to East African peanut meal, then to a fungus on the meal itself (Aspergillus Flavus). The toxic principle was then isolated (aflatoxin) and shortly afterwards Brazilian peanut meal was similarly incriminated. Within about six months of the original reports of the disease being received, poultry organizations all over the world were quickly notified about these toxic meals with immediate benefit to the industry. These fungus diseases, or mycotoxicoses as they are called, may be emerging as a threat to livestock and a new field of research is being opened up. In New Zealand, for example, facial eczema of sheep has been proved to be due to a fungus on rye grass, and in Canada the fungus aspergillus found in moldy grain and bedding, is

apparently infecting poultry and humans. Research into pulmonary emphysema of cattle is also exploring a fungus as a possible cause.

Before passing on to a more detailed review of the epizootiology of foreign poultry diseases, mention must be made of the fact that several poultry diseases are readily transmissible to poultry workers and veterinarians engaged in eradication work in epizootic areas. The highly infectious Newcastle Disease has frequently caused fever and severe hemorrhagic conjunctivitis in humans in Africa, and there are cases also of human ornithosis from African grey parrots, parakeets, canaries, and budgerigars. The latter cases, however, generally occur where domestic cage-birds are purchased from infected pet shops. There is little evidence that avian tuberculosis, salmonellosis, and pox infect man directly and listeriosis, although readily transmissible to man from poultry, is rarely encountered in the field.

I would now like to pass on to some of the more important poultry diseases starting with Newcastle Disease. This disease was named after its discovery in 1926 at Newcastle in England (not at Newcastle in Wyoming). It reached East Africa from Asia by 1935 and is now found all over the world. In the USA it has smouldered since 1944

and is relatively mild in nature, whereas overseas the virus is velogenic, spreading rapidly and of high virulence. In Britain the presence of velogenic virus has led to a policy of quarantine and slaughter, with the payment of indemnity to poultry owners, combined with vaccination. In Africa, the virus is also velogenic, but shortage of finance precludes a policy of quarantine, slaughter, and indemnity. There the disease has no seasonal incidence, whereas in the USA outbreaks appear to be more common in the winter, generally appearing as the respiratory form and spreading by air-borne droplet infection or by nasal droplet infection from water, feed, and bedding, particularly where houses are overcrowded, badly ventilated, and poorly isolated. In Africa, the disease more commonly spreads by ingestion of contaminated media rather than by inhalation of droplet infection and there outbreaks generally start in backyard flocks, frequently those near sea ports and cities, thence spreading out along the major roads and railways. It should be noted that the disease does not, apparently, spread through the egg to the chick as the virus kills the embryo at about 10 days and only clean eggs can hatch viable chicks. During 1945 undressed carcasses from the USA spread the disease to air bases in Britain, and in South Africa infection entered

via the seaport of Durban from unboiled ships waste containing poultry scraps. The traffic in African grey parrots from the Belgian Congo to Nairobi in Kenya some 2,000 miles away, is another instance of the many and varied ways in which this disease spreads. A most unusual, if not amusing outbreak, was reported from Leopoldville in the Congo in apparently well-isolated flocks owned by Europeans. The outbreak remained a mystery until it was discovered that African natives were throwing their hens, tied by a string, over the European's fences at night to be hauled back at dawn, after a free insemination by the European-owned pedigree Rhode Island Red Cockerels! Newcastle Disease is very easily spread from packing plants by personnel, equipment, and carcasses and there is also a considerable risk in purchasing replacements during outbreaks. There are no proved cases in Africa of birds recovering from natural infection to become carriers of the disease. The virus has been recovered from bedding and from the feces of dogs and cats feeding on carcasses, and is generally very resistant. The virus has been recovered from chilled chicken lung 17 months after infection, from carcass bone-marrow up to four months, and from infected premises in winter up to seven weeks. In Africa the rapid spread and high mortality of

Newcastle Disease combined with the poor control of movement and the impossibility of adequate quarantine enforcement has left vaccination as our main weapon against the disease. The difficulty in using live vaccines lies in the danger of their precipitating other infections such as chronic respiratory disease. The latest vaccine is beta-propio-lactone inactivated vaccine. Broilers are vaccinated at three weeks of age and layers are boosted at 15 to 20 weeks, thereafter annually between laying periods. BPL can be combined with other vaccines, does not confuse the diagnostic hemoglutination inhibition test outside one month after vaccination and it is cheap, easily stored and handled. Chickens born from vaccinated hens appear to have a reasonable immunity until three weeks of age. Live vaccines are still in use in Africa as they are easier to administer than dead vaccines, being given in the form of sprays, dusts, or in the drinking water. However, a team of two trained catchers and four vaccinators can inject 4,000 birds an hour with the dead BPL vaccine. Live vaccines are very liable to produce birds excreting virus for anything up to four weeks after vaccination.

The control of Newcastle Disease also depends on efficient depopulation of infected premises for at least one month, area quarantine for for two months, and thorough cleansing and disinfection, using two per cent caustic soda with strict carcass disposal, boiling of waste food fed to poultry, and the control of visitors and poultry products moving through plants and farms. The disease can apparently spread by every possible route and is therefore extremely difficult to trace. For example, swarms of butterflies in the tropics have been considered responsible for disseminating the disease.

Epidemic tremors (infectious avian encypholomelitis) is fairly common in overseas territories. It was first recorded in the USA as early as 1932. The nervous symptoms can easily be confused with Newcastle Disease, crazy chick disease, and fowl paralysis so that laboratory examination is generally necessary for accurate diagnosis. The symptoms are generally only seen typically in chicks under six weeks of age, older birds rarely showing symptoms even when infected. The affected birds are dull, unable to stand, and fall about as if paralyzed, collapsing on their sides. A drooping of one or both wings is commonly seen, and, if the bird is held in the hand, a faint trembling of the muscles of the head, neck, or body can be detected.

The cause is now known to be an enterovirus, Erro gallinae, 20 to 30 microns in diameter, which is passed in the excretions of recently infected birds. The disease spreads by the ingestion of infected droppings and contaminated litter of food. The epizootiology of this disease differs from other poultry infections in that previously infected and recovered pullets give an extremely high and solid immunity to their chicks, protecting them absolutely up to six weeks of age. In contrast, chicks bred from non-immune parents are highly susceptible for the first three weeks of life, showing clinical symptoms around 10 to 14 days after infection. Infected chicks can infect susceptible chicks if they are in direct contact, as in battery brooders. An infected pullet can pass the virus through her ovaries thence to the eggs, producing infected new-born chicks which often die within the first week, meanwhile infecting susceptible in-contact chicks. It is not known whether recovered birds are carriers or not, but it is believed that chronic carriers do not exist. Summarizing, the stages in IAE are therefore:

1. Susceptible pullets produce susceptible chicks.
2. Infected pullets produce infected chicks.
3. Infected chicks infect susceptible chicks.
4. Recovered pullets produce resistant chicks.

The virus spreads slowly and flocks generally show all phases of infection, viz., susceptible, infected, and resistant birds. Growing stock which are infected between the age of 13 weeks and point-of-lay show no symptoms, but, when they reach point-of-lay, there is a drop in fertility and a lowered egg production. As brain and nervous tissue rapidly decompose after death, diagnosis is best made by sending live sick chicks to the laboratory for microscopical examination of brain and nervous tissue. Also, blood samples can be sent for the serum-neutralization test. Some laboratories are now using the embryo susceptibility test, using a field virus adapted to eggs injected into fertile eggs. Eggs from susceptible parents show degeneration of the embryo around the 17th day. There is no treatment known for this disease. The control measures consist in hatching chickens from flocks which are known to be immune, and in vaccinating growing stock with a live virus, and laying stock with a dead virus. It is not necessary to vaccinate all the growing stock with live virus as a selection of vaccinated growers will spread the disease to the in-contacts. Immunity after live virus infection is believed to last for about 18 months.

The fowl leucosis complex is rather rare in native poultry overseas, but in the European breeds, particularly the Rhode Island Red, the

incidence appears to be increasing, particularly in modern units where birds are 'forced'. The disease is not only seen more frequently, but it would appear to be occurring at a much earlier age than it did 10 years ago, sometimes appearing as early as three weeks of age and killing 50 per cent of the chickens by the 10th week. This is popularly called 'galloping leucosis'. The fact that American research workers have developed strains of breeds with increased resistance to this disease supports the theory that breed susceptibility does exist in the field and there does appear to be some age resistance in adults. There is some conflict on whether this disease can be directly transmitted through the eggs to chicks. It is possible that research workers in the USA who have isolated the virus may in the near future produce an avianized vaccine, which will be a tremendous step forward in the control of leucosis.

The next two diseases CRD and Infectious Sinusitis are generally associated with the Mycoplasma gallisepticum organisms and in South Africa severe outbreaks, with a death rate up to 40 per cent and a morbidity of 90 per cent with marked growth depression and reduced egg production have been recorded. The disease spreads slowly through flocks over a period of several months, leaving in its wake carrier

birds and highly infected eggs. The natural immunity is weak and, so far, no reliable vaccine has been found. Hatcheries may increase the dissemination as infected eggs may appear normal and expose the entire hatch. Antibiotic dipping of eggs is being tried at some hatcheries overseas, but its use in America is considered impracticable at present. If a flock is small and well isolated, treatment of infected birds may be adopted with the object of lowering the infection, but it is generally safer to cull rather than to treat infected birds. Britain has a recently developed drug reputed to cure infected birds with one injection and the possibility of sterilizing eggs by injecting the parent birds is being explored. Overseas, streptomycin has proved reasonably effective but tends to produce resistant strains and can be toxic to young poults. When seriologically positive birds are disposed of beforehand, the treatment of small flocks using streptomycin in micronized dustguns has held the disease in check. In the tropics, the results of serum or plasma plate tests are often difficult to read in extremely hot weather under field conditions. An economical method of testing is to select at random about 30 birds at the start of lay and if these birds appear negative, to bleed 10 per cent of the remainder.

In the wet, humid areas overseas, coccidiosis is still serious. The aim is not to cure and thereby sterilize birds leaving them open to reinfection, but to create an immunity. As the numerous drugs on the market vary greatly in their curative efficiency on each different species of coccidia, there is a risk that the drug will fail to reduce infection and that drug-resistant strains will emerge unless the species of coccidia is accurately identified before treatment starts. Immunity can best be achieved by intermittent treatment with the appropriate drug at intervals of two to three days. The latest method of using a mixed vaccine of oocysts in the drinking water has not yet been proved reliable in Africa. It must be remembered that immunity to cecal coccidiosis in chicks does not provide them with immunity against intestinal coccidiosis as adults. Indiscriminate treatment with the sulfa drugs also carries the risk of causing a highly fatal hemorrhagic syndrome. The addition of 8,000 IU of vitamin K per pound of food is recommended to aid recovery and to reduce the severity of infection.

Black head in turkeys is an example of a disease which has greatly increased in virulence overseas during the last few years. As the vector worm egg has a tough shell and persists for a long time in the soil, continuous preventative medication must be adopted.

Infectious synovitis is not yet very common and a rapid cure can be effected by terramycin in the drinking water for five days.

Sporadic outbreaks of fowl cholera do occur but not so commonly as in the eastern USA. The prevention is by sulphaquinoxaline in the drinking water, by Avisepticus gallinarum bacterin or by avianized vaccines. Treatment is by streptomycin injection or by choloramphenicol in the feed.

At present infectious laryngo-tracheitis is not very common in the tropics, but it is a serious disease in the colder countries overseas. It requires drastic control measures, as it spreads rapidly and causes sudden death or extreme emaciation. There is a reliable avianized vaccine available, applied to the cloaca of birds at six weeks to four months of age. It may be that ILT will steadily increase in Africa as large units become more numerous.

Fowl pox is very common in Africa and Asia and its spread is aided by mosquitoes, particularly at the beginning of the rainy season. It attacks adults, causing loss of weight, drop in egg production, and dullness over a period of about a month. The disease is slow-spreading and generally produces typical warts on the comb and wattle or diptheritic membranes in the mouth. A rapid field test is to grind up the warts in buffer solution and apply to the feather follicles,

producing a reaction in about a week. In the laboratory, Bollinger inclusion bodies can be demonstrated in tissue specimens. Vaccination affords highly efficient protection, for layers pigeon pox vaccine is used and for growers, fowl pox vaccine.

In tropical countries where argasid ticks are found, losses can be from 50 to 90 per cent in untreated birds attacked by spirochetosis. The cause is spirocheta gallinarum, a spiral organism staining readily with Giemsa. Fowls, ducks, geese, turkeys, pheasants and partridges are all highly susceptible to this disease. The usual vector is Argas persicus, the common fowl tick, but the disease can be spread mechanically by lice, stable flies, and mosquitoes and sometimes by fowls eating infected ticks or infected tick eggs. Fowls of all ages from 10 days up are susceptible. The acute form of the disease produces severe anemia, persistent green diarrhea, thirst, high fever and paralysis, whereas the sub-acute form shows marked jaundice and emaciation. On post-mortem, the hugh, swollen spleen, up to six times normal size, is almost pathognomic and the carcass is generally anemic or jaundiced, with hemorrhagic enteritis. The diagnosis is by Giemsa-stained blood smear from a sick bird. It is generally useless to send blood from dead birds as the spirochetes usually die and are difficult to find two hours after death. Treatment

is by a single injection of five to ten thousand units of penicillin. The arsenobenzol group of drugs is also useful for the sub-acute form, but one must beware of toxicity. Both recovered and treated birds are thoroughly immune. In Australia, the disease is well controlled by regular annual vaccination of three to four months old birds with an avianized formol-killed culture vaccine. This vaccine can be used during outbreaks, as immunity will develop within three days of injection. In endemic areas poultrymen tend to treat all purchased birds with penicillin, if any of them show signs of illness, on the presumption that spirochetosis is the cause. It should be noted that the adult argasid tick can survive for two years without a blood meal and that infection with spirochetes can pass through the egg of the tick to all stages in the tick life cycle. The removal of fowls for periods as long as two years is, therefore useless as an eradication measure. These ticks are three-host ticks which, apart from the larvae, feed on the birds at night. The control measures advised are to eradicate ticks and provide tick-proof quarters for the birds.

There is a group of diseases of poultry overseas resembling malaria in the human being (plasmodium infections) which cause severe losses in birds imported from the USA or Europe. They are mosquito-borne diseases of the blood and it is believed that the true host is

the wild jungle fowl. Local indigenous birds appear to have a natural resistance to these diseases, but imported pedigree stock are extremely susceptible, showing severe anemia and pericarditis. Quinine is a highly effective treatment.

On some farms overseas, a chronic sporadic wasting disease of adult fowls occurs on some farms year after year. This disease is listeriosis and on post-mortem, birds will show massive necrosis of the heart muscle and focal necrosis of the liver. Mild cases are easily cured by penicillin, others respond to the tetracyclines. No guaranteed vaccine is available for this disease and very little is known of the control measures that should be adopted.

In Africa and Asia, fowl typhoid is a very common disease in native ranged and backyard flocks, but it is less common in modern hygienic units such as batteries, etc. It can affect young chickens and produce a 70 per cent mortality over a two-week period, but it is also common in birds over two months of age where it appears as a less acute disease. A combination of vaccine and treatment has been employed with some success, but the creation of resistant strains of organisms led to the withdrawal of this technique in many areas. Infected birds can be detected by laboratory tests and in Africa specimens of long bones (femur) are generally sent from the field for

bone-marrow tests while live birds are tested by the whole blood test at the premises. After the removal of the reactors, the remainder of the birds are moved to clean premises and given 10 days of furozoladine treatment. This, as stated, unfortunately produces carriers and a switch can be made to terramycin which, although it cures birds, appears to leave them sterilized of infection and susceptible to reinfection. This produces persistent antibodies in many of the flocks which confuses subsequent diagnostic tests. Two vaccines are in general use, firstly, a live rough strain given at eight weeks of age which produces a reasonable immunity of four months duration. This vaccine suppresses mortality but will not stand up to a heavy challenge and it interferes with the blood test. Secondly, there is a vaccine for adult layers which is a live smooth strain 1909, but the incubation of eggs from vaccined birds has to be prohibited for six weeks after vaccination. In general it can be said that the vaccination policy overseas is meeting with varying success, but a quarantine and slaughter policy is not feasible under the economic conditions prevailing in the many of the under-developed countries. Another difficulty is that adult birds, while carriers, will rarely show clinical symptoms. A routine worthy of adoption is to trace back from the farm to the issuing hatchery, all batches containing dead chicks on arrival.

In recent years some diseases of livestock overseas have been emerging as economically important entities, such as Lumpy Skin Disease of cattle, Bluetongue and African Swine Fever. One such "emerging disease" of poultry has recently been reported in Japan and Burma and all veterinary services have been asked by the Food and Agricultural Organization to maintain a careful watch for the disease in their areas. The disease is caused by Leucocytozoon caulleryi, a blood protozoon, and is an acute disease clinically resembling fowl cholera. It affects birds generally at 3-5 months of age with mortality rate of about 20% in affected flocks. Birds may die suddenly with no obvious symptoms or may cough up ropy, blood-stained mucous from the respiratory tract, or pass blood from the cloaca. Autopsy reveals hemorrhages in the lungs and kidneys, with greyish white foci (collections of megaloschizonts) in the viscera, meninges, mucosae, serosae and the subcutis. Diagnosis is by Giemsa or Romanowsky-stained blood smears demonstrating gametocytes and merozoites, and by low-power examination of crush smears of small portions of lung or kidney. Differential diagnosis excludes fowl cholera (by crush smears of hepatic lesions), pasteurella multocida (by blood smear), Eimeria necatrix infection (by examining intestinal lesions with hand lens). The disease in Japan is transmitted by the

Ceratopogonid fly, Culicoides arakawae. The control of this fly vector which breeds on paddy fields and marshland is not yet feasible and no sound recommendations for the control of the disease can be made at present. No effective drug has been found to control or cure the disease.

In conclusion, it can be stated that the Veterinary profession overseas is becoming increasingly concerned with the control and eradication of poultry diseases, and that the poultry industry overseas is playing an increasing part in providing animal protein for the under-developed areas of the world.

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